

VOL. XLIV

NUMERO 10

Boletín de la Asociación Médica de Puerto Rico

ORGANO OFICIAL



PUBLICACION MENSUAL

OCTUBRE

1952

Entered as second class matter, January 21, 1931 at the Post Office at San Juan, Puerto Rico, under the act of August 24, 1912.

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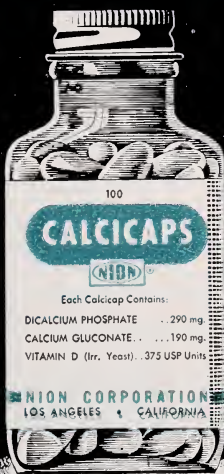
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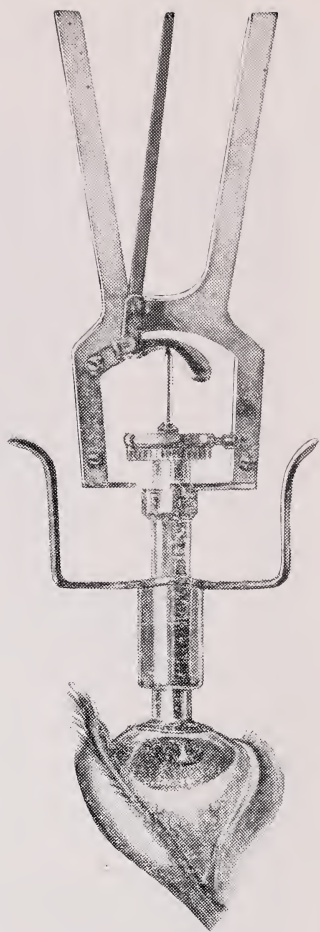
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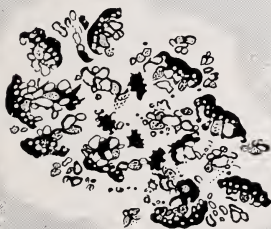
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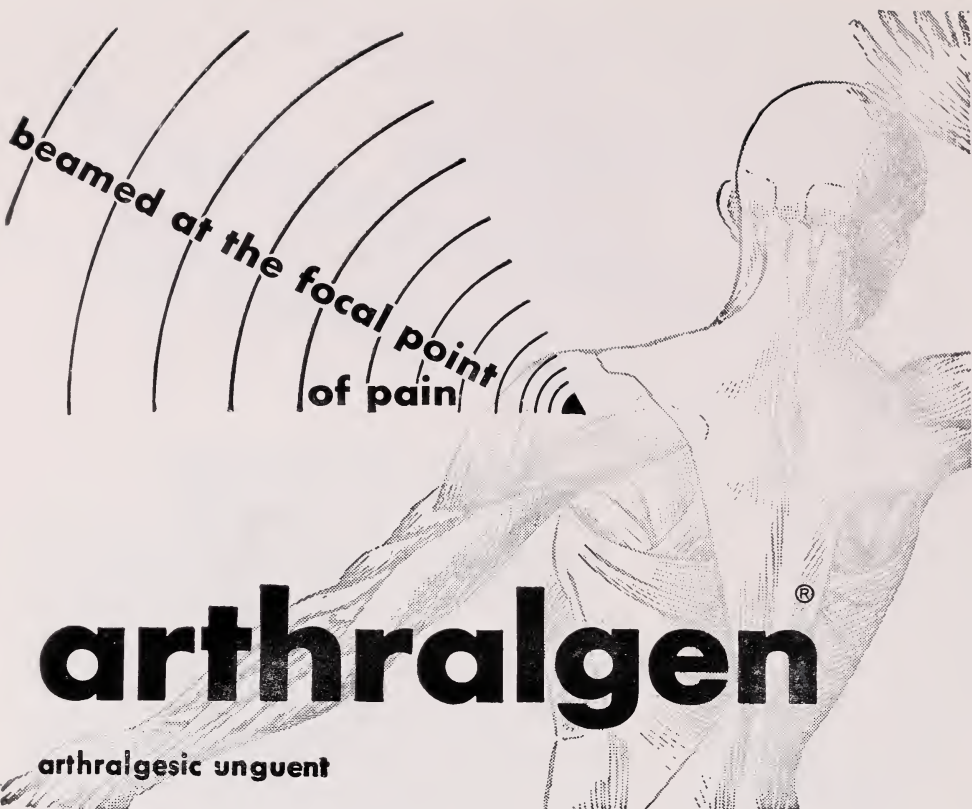
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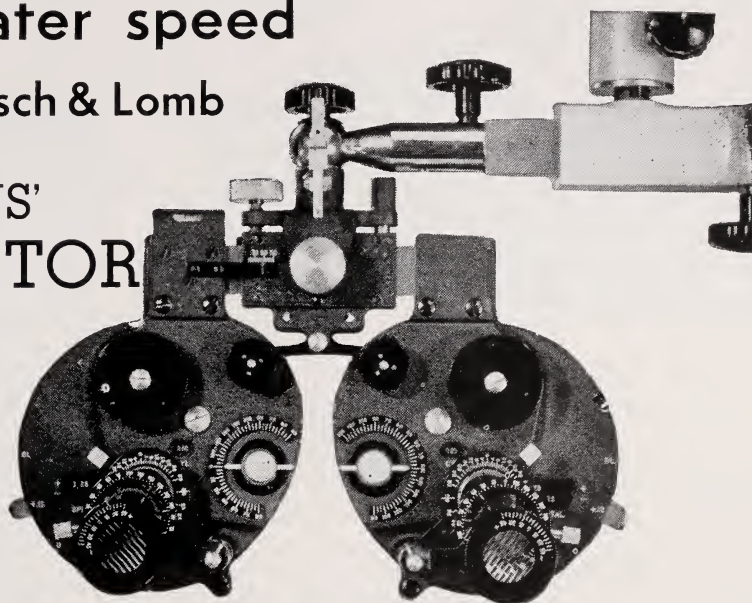
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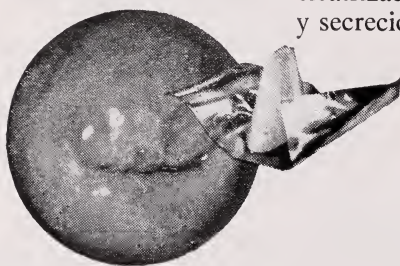
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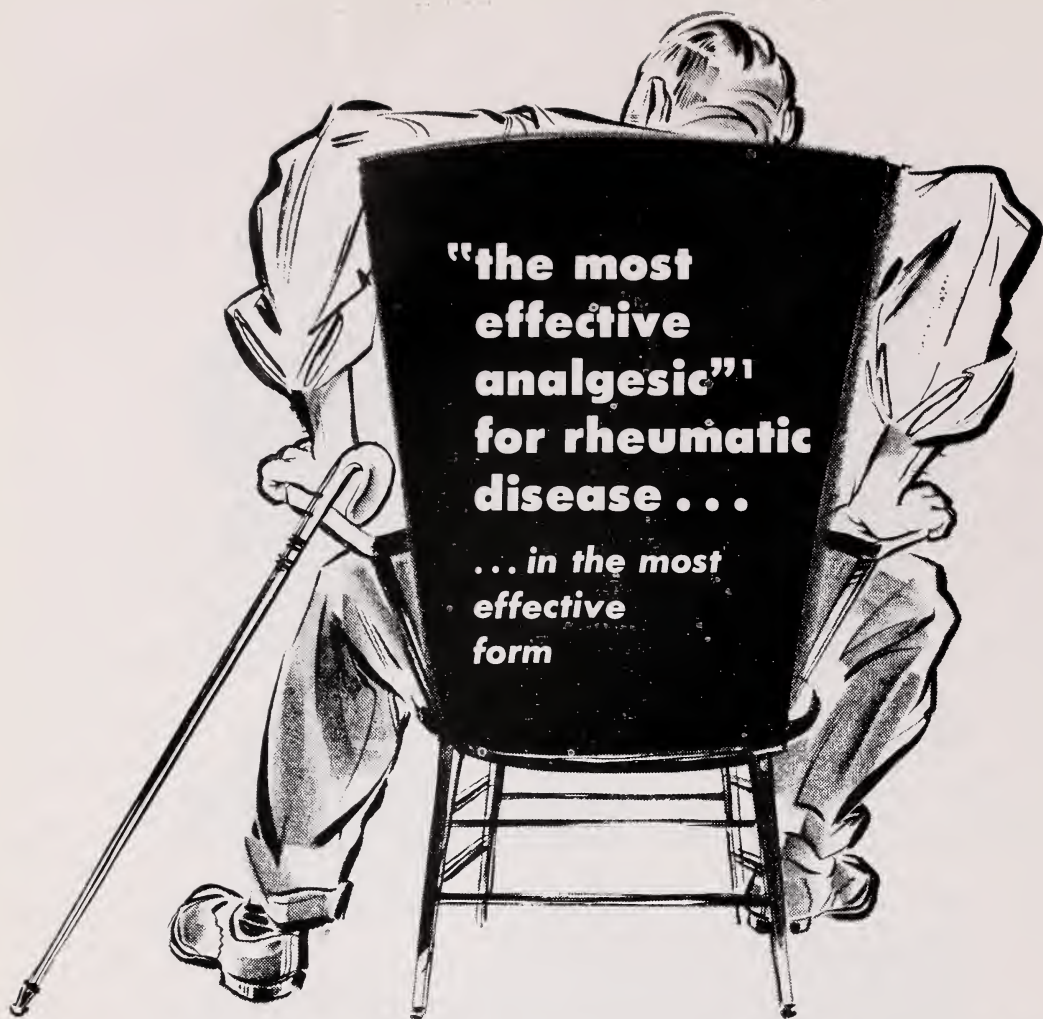
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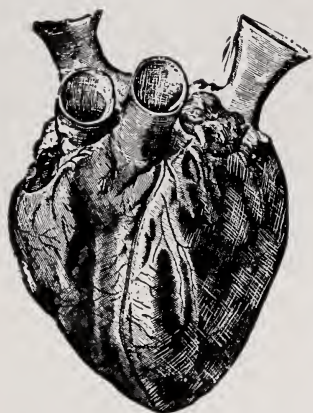


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*Blake, F. G., Friou, G. J., y Wagner, R. R.,
Yale J. Biol. & Med. 22:495 (Julio) 1950.

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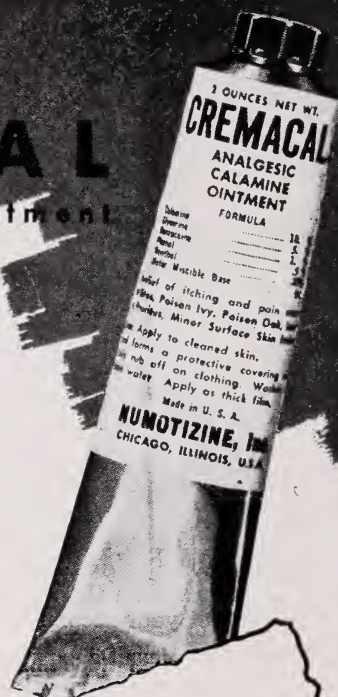
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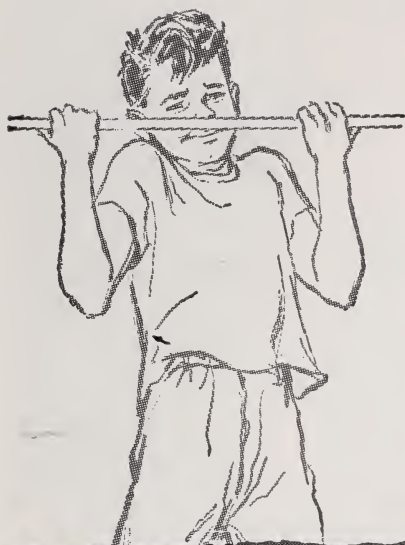
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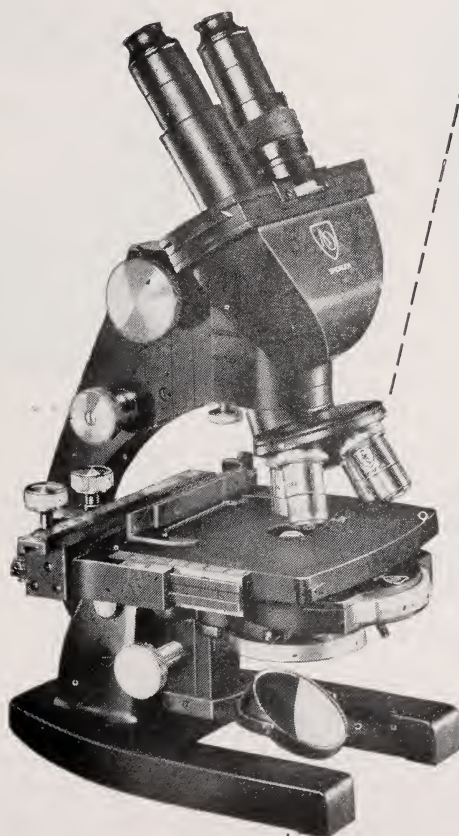
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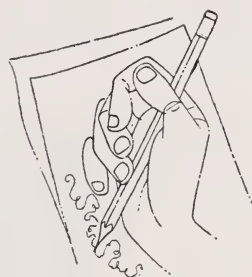
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ASOCIACION MEDICA DE PUERTO RICO

VOL. XLIV

OCTUBRE, 1952

No. 10

STUDIES OF HOOKWORM DISEASE IN PUERTO RICO

PRELIMINARY REPORT

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and

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San Juan, Puerto Rico

Among the causes of death in Puerto Rico,¹ ankylostomiasis occupied the thirteenth place, with a death rate of 47 and 34 per hundred thousand population, having been responsible for 2.1 and 1.8% of all deaths occurring during the years 1933 and 1934, respectively. These figures indicated a reduction in the mortality from this condition compared to that of the previous ten years when the mean death rate was 82 per 100,000 population. The control and sanitation campaign instituted by the Insular Health Department with the cooperation of the International Health Board of the Rockefeller Foundation has significantly contributed to reduce this mortality. The death rate has continued to fall and in the year 1950, the reported death rate for ankylostomiasis in Puerto Rico was 0.9 per 100,000 population.* In spite of the economic depression, unemployment and undernourishment so prevalent among the rural white population of Puerto Rico up to the year 1933, when government relief and welfare measures became widespread throughout the Island, the pale-faced, apathetic and indolent "jíbaro" or mountaineer is no longer as frequently encountered as in the days of Colonel Ashford and the Puerto Rico Anemia Commission, when at the turn of the century, it was estimated that 90% of the rural population was suffering from this condition, and one-

* Personal communication, Bureau of Vital Statistics, Department of Health, San Juan, Puerto Rico.

third of the Island's inhabitants received treatment against the hookworm.

But hookworm disease, accompanied by severe anemia and chronic invalidism, is still commonly encountered in certain localities in the mountainous interior of Puerto Rico. Among a group of forty-five young white males who in 1933 sought anthelmintic treatment at the local health unit in the town of Lares, a former coffee center for the central western part of the Island, the highest red cell count and hemoglobin were 3.6 millions and 72 per cent (10.4 grams) respectively.

Nine white males seventeen to twenty-five years of age, harboring heavy infections and presenting intense anemia, were carefully selected from this group and comprise the basis for the present report. Upon being hospitalized these individuals were apparently free of other disease conditions.

It was the purpose of this investigation first, to provide hospitalization for these individuals in order to determine the anemia present before instituting therapeutic measures, and to obtain other clinical and laboratory data important in hookworm disease; second, to test out the effect of daily doses of ferrous salts, of liver extract, and of a well balanced diet on the anemia and well being of the individuals studied, before and after the removal of the worm burden; and third, to determine the end results of therapeusis, one to several years after discharge from the hospital and their return to the environmental conditions prevalent before hospitalization.

Three hundred and twenty determinations of peripheral blood morphology, including hematocrit readings and sedimentation rates, have been performed during a period of seven years comprising the study. Report on other clinical and laboratory data will be the subject of a future communication.

Stoll egg counts were determined from samples of twenty-four hour stools from each individual during hospitalization. Egg counts varying from 25,000 to 134,000 eggs per gram of feces were found. Upon anthelmintic treatment, worm counts were performed in all cases.

All patients remained in the hospital for a minimum stay of 50 days and a basic diet low in iron was allowed.

On admission to the hospital and during the period of observation prior to employment of therapeutic measures, the red cell count and hemoglobin ranged between 2.2 to 4.0 millions, and from 27 (4.0 grams to 51 (7.4 grams) per cent. The anemia encountered was of the microcytic and hypochromic types in all individuals.

Following the administration of ferrous salts (iron sulfate 1 gram daily; iron ammonium citrate 50% solution, 12 cc. daily)

and a full hospital diet over a period of 30 days prior to the removal of the worm burden, the blood response and the change in general condition of the patients was quite similar in all cases, irrespective of the therapeutic agent administered. There occurred a gradual but persistent rise in blood values, until a certain level was obtained, after which no further significant change was observed. However, the clinical improvement in all cases was more marked in those who received iron.

Liver extract parenterally administered, containing the Cohn fraction and potent in sprue and in other tropical macrocytic anemias, produced no appreciable change in blood values or in clinical condition in two individuals. After a 30-day trial under liver therapy, iron salts were administered to these before leaving the hospital.

Within ten days after the administration of an anthelmintic, the red cell count rapidly rose to normal limits, in some cases ranging from 4.69 to 6 millions; in others the rise was gradual and slower requiring three to four weeks. Hematocrit values also were found within normal figures. Sedimentation rates were with few exceptions within normal variations. The hemoglobin, however, did not follow the rapid rise of the redcells, but lagged somewhat behind, ranging from 70 to 100% (10 to 14.5 grams). The number of adult *Necator americanus* passed varied from 1063 to 2979 for each individual.

Upon discharge from the hospital, the improvement in physical and mental condition of the patients was as striking as the increase that had taken place in the blood values. After leaving the hospital and returning to their home environment, all treatment, was stopped. During the subsequent four months, hematological studies revealed that the hemoglobin had gradually risen to normal figures, the red cells remaining within the limits of normal variation.

During the first year after hospitalization all individuals were found to harbor light hookworm infections, the excreta being positive to the Willis flotation method only. Towards the end of the first year, all but one case were enjoying good health with no anemia. Nineteen months after hospitalization the case that was observed to be slightly anemic and in fair health by the end of the first year, died from pulmonary tuberculosis. Observation of this case during terminal illness was not possible. The condition was not demonstrable by X-ray examination of the chest during hospitalization. The last stool examination, performed 10 months before death, revealed few hookworm ova. During the second and third years following treatment, two other individuals showed moderate anemia, having lost about 30% of their red cells and

hemoglobin present on discharge from hospital. Egg counts varying between 400 and 10,000 eggs per gram of feces showing higher infections than during the first year, were observed in all cases.

During the seventh and last year of follow-up study, another individual died due to unknown cause, as close observation prior to death was not possible. Blood studies performed about one year before death, however, revealed values as low as when first observed seven years previously. The stools contained 60,450 hookworm ova per gram of feces. In view of such findings, it is believed that death may have been caused by hookworm disease. The remaining seven cases were living and actively at work. Four appeared quite healthy and were not anemic but three others presented evidences of moderate anemia. Regarding stool examination, all but one of the cases presented heavier hookworm infections than those observed prior to treatment seven years before.

The results of anti-anemic and anthelmintic treatment were, however, still manifest in all the surviving individuals. Instead of the former semi-invalids who for several years had been unable to perform a complete day's work and who were dependent upon their families for support, there is now a group of active hard-working, self-supporting, agricultural laborers. Strangely enough, this physical and mental rehabilitation had not enabled them to earn higher wages, but personal hygiene has been greatly and permanently improved. The moderate anemia already present in three cases and the parasitism found in all has not as yet impaired their renewed vigor and vitality.

Notwithstanding the smallness of this group of individuals, certain inferences can be made from this medical, public health, social and economic problem that still affects thousands of Puerto Rico's rural population.

With Suárez^{2, 4} Rhoads, Castle³ and associates, it is believed that ferrous salts in adequate daily amounts constitute the drug of choice for the relief of the anemia of hookworm disease. However, we have observed that iron alone is insufficient to produce a complete cure of the anemia without the removal of the worm burden and only when an effective anthelmintic is administered, do the blood values rapidly rise to normal figures.

It has also been shown in the group studied that sometime between the end of the first and third year after the cure of the anemia and removal of the worm burden, a moderate anemia in some cases and an increased parasitism in all are again evident. Now, if this is true of individuals who have received adequate anti-anemic therapy and vermifuges have been given under controlled conditions, what can be expected to occur in those thousands of cases which are given an anthelmintic, a handful of Blaud's pills

and told to return to their homes? Does deworming constitute a cure for the anemia that is beyond all doubt the underlying cause of their misery and invalidism? The present study indicates that about one month of iron therapy is required to raise the red cells and hemoglobin to normal values, after deworming has been accomplished. Is, then, this incomplete treatment justified in view of its apparent deficiency?

The diet of the average Puerto Rican rural laborer has been found to be deficient in animal proteins. A study of the diet in this group before and after hospitalization confirms the above statement. It is the opinion of the authors that the anemia found in heavily infected individuals is not entirely produced by the parasites, but rather is the result of a combination of prolonged inadequate diet prevalent since childhood and an early acquired and continued parasitism. The comparatively slight infection accompanied by moderate anemia observed in two of our cases, three years after treatment, appears to support this contention.

In the opinion of the current authors, the problem of hookworm disease in Puerto Rico is mainly an economic one and no form of treatment, whether mass (anthelmintic) or anti-anemic therapy, is the final answer.

In conclusion, observations on this small group of individuals seem to indicate that while intensive iron therapy, a well-balanced diet and an effective anthelmintic were responsible for a rapid recovery from the chronic anemia of hookworm disease, a permanent cure is prevented by the return to the prevailing environmental and economic conditions existing prior to hospitalization and treatment of the individuals studied.

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A REVALUATION OF THE SIGNIFICANCE OF ALLERGY IN TUBERCULOSIS*

JOSE A. AMADEO, M.D., F.C.C.P.**

The significance of allergy in tuberculosis, a very significant question in a very significant disease, remains controversial. Hence, any humble attempt, even without additional experimental data, to bring harmony out of discord, may be justified.

The preponderance of allergyphiles over allergyphobes among contemporary experts in tuberculosis, shows that Krause's dictum, "Allergy is a function of immunity", still dominates phthisiology. This truism so powerfully expressed by a figure of such towering stature, has retained supremacy and has exerted a profound influence on all thought concerning allergy in tuberculosis, during the last quarter century.

As a "function of immunity", and as the only available indirect measure of immunity in tuberculosis, it is difficult indeed to think of allergy in relation with tuberculosis, except as something good, since after all, immunity is always the maximum desideratum in every infectious disease.

Furthermore, one is always aware of the intimate interrelationship between allergy and immunity in tuberculosis and of their indivisibility, by always remembering the total loss of allergy that accompanies, the total loss of immunity, during the exhausted, unreactive, preagonal stage of the disease.

And still more, it is always foremost in every mind, that allergy by its power of focalization, of fixing bacilli in situ, safeguards against their spread to the meninges, still one of the most dreadful catastrophes in medicine.

No wonder then that a tag inscribed "nolli me tangere" has attached itself so strongly to the thought of allergy in tuberculosis with the resulting lag in research along this line. And no wonder that the large group of allergyphiles comprising the distinguished exponents and champions of the monumental prophylactic work with the bacillus Calmette Guérin vaccine, are searching for higher allergy producing vaccines, under the assumption that the best vaccine is the one which produces the highest degree of allergy for the longest period of time; and are looking for the highest natural resistance to tuberculosis among families with the highest allergic diathesis.

* Read before the P. R. Chapter of the A.C.C.P. at the 1951 annual meeting of the P. R. Medical Association.

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But tuberculosis is the paradoxical disease par excellence, and to any student unwilling to side-track in his mind the different appalling paradoxes that he encounters in tuberculosis, allergy will reveal itself in quite a different light.

Pathology classifies tuberculosis as a chronic infectious disease. As such, the responsible mycobacterium tuberculosis per se, should only give rise to chronic, mild, local lesions with chronic, mild, focal and constitutional manifestations and with great tendency to spontaneous healing. At least among tubercle bacilli, increased virulence only means increased power of survival with increased opportunity for multiplication, obtained through increased virulin, the specific constituent of virulent bacilli which resist opsonic action. It does not mean any increase in the destructive power of the weapon of each individual bacillus. It does not mean that the more virulent strains of tubercle bacilli will produce a more virulent toxin with increased local destructive power. The tuberculotoxin obtained from the most virulent tubercle bacilli has exactly the same local toxic power as that obtained from the least virulent strains. Therefore, increased virulence of tubercle bacilli even when associated with decreased resistance of the host, can only account for a quantitative increase in the number and size of the chronic, mild, local lesions produced in the host but can not account for any qualitative alteration in the pathologic characteristics of each one of these more numerous and extensive —chronic, mild, local lesions, since tuberculotoxin, the only weapon of each individual tubercle bacillus, is never altered and always remains a constant, mild, local irritant, incapable per se, of inducing any acute local inflammatory reaction and, of course without any direct caustic or necrotizing power.

And yet, in practice, in the majority of the cases, instead of these chronic, mild, local lesions, with chronic, mild focal and constitutional symptoms and with great tendency to spontaneous healing, paradoxically, we encounter exactly the opposite pathological and clinical pictures, with acute, severe, local inflammatory reactions that give rise to acute, severe, exudative infiltrations progressing to necrosis and ulceration and accompanied by acute, severe, focal and systemic manifestations and with very little tendency to spontaneous cure.

Since the firing power of the only weapon of each individual tubercle bacillus, the only etiologic agent in tuberculosis, is never altered, and never increases from that of a "toy-gun" to that of a real gun with direct local destructive power, we have to look for an added factor to be able to get a logical explanation for this paradoxical metamorphosis of a mild, chronic disease with very little

killing power, into a severe, acute disease with tremendous killing power.

The great Myers gives a masterful description of the presence and action of this added factor in his also famous dictum, "Allergy is the indispensable ally of the tubercle bacillus as a destructive agent. In the absence of allergy, tubercle bacilli are practically harmless to man, but when allied with allergy which it produces, it takes the first place among diseases as a destroyer of tissues and organs".

But, a false impression may be created by the natural tendency to reduce this dictum to its obvious, simplest formula:

(a) Tuberculotoxin sine allergy = mildest poison.

(b) Tuberculotoxin + allergy = deadliest poison.

To insure a clearer conception, the above formula must include the expression of the unalterable, constantly mild character of tuberculotoxin as a local irritant, and must be re-written to read:

(a) Tuberculotoxin sine allergy = mildest local irritant — the mildest poison.

(b) Tuberculotoxin + lots of allergy = the same, unaltered, mildest local irritant but with terrific **indirect** necrotizing power — the deadliest **indirect** poison.

It is the progressive increase in allergy, the altered state of reactivity of the tissue, induced by repeated sensitizing re-infections, that makes the tissues react more and more violently to a **constantly mild local irritant, with the resulting acute exudative infiltrations becoming more and more intense and causing more and more interstitial distension, until capillary blood supply is obliterated, the tissue is choked to death, and ischemic necrosis supervenes with subsequent caseous degeneration of the tissue already necrotized by ischemia and with ulceration and cavity formation**; the keystone in the differential diagnosis between the primary benign, self healing type of disease, in which with less allergy, less intense exudative infiltrations are reabsorbed before the tissue is choked to death, and only few, small, central nodules among these exudative infiltrations may undergo ischemic necrosis with subsequent caseation, but with organization and calcification instead of ulceration; and the malignant, progressive, re-infective disease with extensive ulceration and **cavity formation**; again the keystone of bad prognosis, the focus of continuous endogenous reinfections, the perpetuator of the vicious cycle of constant reinfection and resensitization, with progressive increase in allergy and progressive increase to a hundred fold, to a thousand fold in the **indirect** killing power of the tubercle bacilli.

Thus we see how this added factor, the progressive increase in allergy, with progressive increase in the violence with which

more and more highly sensitized tissue, responds to tuberculotoxin, a constant, unalterable, mild local irritant, gives the only logical explanation to the first great clinico-pathological paradox in tuberculosis, namely: the mycobacterium tuberculosis, a single etiologic agent, with a single mild weapon, producing in each, single, "bi-peps sine pluma", two entirely different diseases, with entirely different pathological and clinical characteristics; always at first the chronic benign self healing disease and always later on, with subsequent reinfections, the acute, malignant, progressive disease.

Obviously, with an extraordinarily massive, lasting, first infection, or with uninterrupted, exogenous reinfections, the disease may progress uninterruptedly to the malignant, re-infective, progressive type, the so-called, "primary progressive disease".

The chronology of these events in the natural history of the disease, constitutes from an immunologic point of view, the 2nd. great paradox in tuberculosis, to wit: always at first, with the rare exception mentioned above, the primary, benign disease, with very little killing power and always later on with each subsequent reinfection, subsequent attacks of more and more severe disease, with more and more killing power, in spite of more and more immunity.

Undoubtedly in tuberculosis there is again, an added factor, intimately interrelated with immunity that acts as a deleterious, cumulative, by-product of immunity, powerful enough to defeat the progressive increase in immunity obtained in tuberculosis as well as in any other infectious disease, by repeated reinfections. Allergy, again can now be easily identified as the only known factor in tuberculosis that fulfills to perfection these requisites. Thus, the deleterious, cumulative effect of allergy, overpowering the increasing, beneficial, protective effect, of a progressive increase in immunity, obtained through natural vaccination by repeated reinfections, again gives the only logical explanation to the 2nd. appalling, immunologic paradox in tuberculosis, namely: each successive bout of the disease, more and more severe, with more and more killing power, in spite of more and more immunity.

This defeat of the protection offered by natural vaccination in tuberculosis is absolute. In Puerto Rico, a very representative area, less than 1% of all deaths from tuberculosis are due to the so-called "primary progressive disease", but of course, nobody can ever die of the re-infective type of the disease, responsible for more than the 99% of all deaths from tuberculosis in our island, without having had the benefit of the most thorough and repeated natural vaccination through previous attacks of the disease.

Unquestionably in tuberculosis, natural vaccination is a complete failure and instead of adding protection it only increases

liability to the disease. Hence, any prophylactic attempt through artificial vaccination was regarded with great mental reservation by all fundamentalists. To them, isolation remained as the only unquestionably acceptable method of control. Of course, the standard of one isolation bed per one tuberculosis death per year, proved a big success, but such an expensive one that it could only be successfully applied in very few, very rich, countries.

Only by sheer necessity was the B.C.G. vaccine tried in man. On the basis of favorable statistics the work was expanded. We may say that in the beginning the B.C.G. men valiantly hanged by the thin threads of limited, favorable statistics, but nobody can deny that at present their position has become very secure and that they now hang by the mighty powerful steel cables, of the most extensive, favorable statistics, in the history of medicine. A very secure but still a rather uncomfortable position.

The success of artificial vaccination with B.C.G. vaccine, in contradiction to the absolute failure of natural vaccination, constitutes the 3rd most appalling paradox in tuberculosis. Of course, it is axiomatic, that other factors, nutritional, environmental, etc., being equal, immunity derives in direct proportion to the amount of antigen, and that natural vaccination with unlimited amount of antigen, always gives incomparably more immunity than can ever be obtained with the necessarily limited amount of antigen of every artificial vaccine.

But now we can easily visualize that the paradoxical success of the B.C.G. vaccine may be explained by its weakness and that natural vaccination defeats itself by being too powerful.

B.C.G. vaccine gives less immunity but with less allergy. Less immunity but less hampered by its deleterious by-product. Tubercle bacilli with little allergy remain rather mild fellows capable of being successfully handled by little immunity.

Natural vaccination gives more immunity but also more allergy. More immunity but more hampered by its deleterious, cumulative, by-product. Tubercle bacilli with lots of allergy become the deadliest fellows, the worst indirect killers that can not be successfully handled even with lots of immunity. Obviously once the tubercle bacilli, with the help of a high allergic state, succeed in isolating themselves in the "sanctum" of a focus of ischemic necrosis, or in the walls of its residual cavity, they can no longer be

reached at all by the bacteriolytic or bacteriostatic products of immunity, no matter how high a level of immunity is attained.

Only in their "sanctum cantorum" built for them by allergy and only in tissue already necrotized by ischemia, are tubercle bacilli capable of inducing further caseous degeneration.

So we see that while obtaining the only possible logical explanation for each one of the three great appalling paradoxes in Tuberculosis, allergy reveals itself rather than as "a function of immunity" as formulated by Krause, as **the deleterious, cumulative by-product of immunity that defeats immunity.**

The validity of this point of view may rest not only on its logical fundamentals, but also, on the logical prediction that the B.C.G. men will fail in their attempt to improve their results with higher allergy producing methods, and that they will find instead of the highest, the lowest natural resistance to tuberculosis among families with the highest allergic diathesis, other factors, nutritional and environmental, being equal.

The advantages of this point of view are threefold:

1st.—It relieves phthisiology of the academic stigmata of three appalling paradoxes.

2nd.—It offers academic reconciliation to the B.C.G. men. A more comfortable position from which their monumental prophylactic work may proceed with less controversy, and with greater impetus, but with logical limitations in regard to their use of higher allergy producing vaccines, and of revaccination, before allergy has completely faded away.

3rd.—It may serve as the necessary stimulus for the increase in research necessary to open wide one of the two strong-boxes that keeps an important half of the medical armamentarium of tuberculosis, the **antiallergic**; so far, kept quite closed in contrast with the other strong-box containing the antibiotics and chemical antibacterials, already widely open.

The fundamental, decisive role, played by allergy in the genesis of ischemic necrosis and cavity formation, the keystone of bad prognosis in tuberculosis, leads directly and inescapably to the addition of antiallergic therapy to antibacterial therapy in the exudative phases of the disease. Of course, during the presence of open cavities, while continuous reinfection and resensitization are

taking place both antibacterial and antiallergic therapy can not be of any permanent avail, but after the elimination of open cavities by efficient collapse measures, antiallergic therapy will again find its place side by side with antibiotics and chemical antibacterials.

It is true that allergy and immunity, the deleterious by-product and the beneficial main product, are inseparable parts of the same process and that therefore, allergy can only be reduced at the expense of a reduction in immunity. Of course, it would be therapeutically unthinkable to carry this reduction, "ad absurdum", into dangerous low levels, but it would be just as insensible to allow allergy and immunity to climb to also dangerous high levels where, an unnecessary excess of immunity, is always, defeated by an excessive accumulation of its deleterious by-product. Obviously both extremes are equally dangerous, with identical end-result: death.

The only problem should be to determine the optimum level of allergy and thus, indirectly of immunity, at which the highest percentage of patients shows the greatest tendency to improve under close observation and this could be easily obtained by doing quantitative determinations of tuberculo-sensitivity, routinely among all patients in several institutions. At this optimum level of allergy and immunity, both allergyphobes and allergyphiles, will move their tents from their separate camps near each extreme and will settle permanently in happy reunion.

Only when this optimum level is clearly determined, could the ultrapotent cortisone and ACTH be used as antiallergics in tuberculosis, with full confidence and without fear of over shooting the mark into dangerous low levels.

The additional effect of ACTH and cortisone of interfering with collagen and connective tissue proliferation, need not be an absolute contraindication for their use as potent antiallergics in tuberculosis. The therapeutic desideratum should be to favor the reabsorption of exudative infiltrations before the tissue is choked to death and the necessity arrives for walling-off with connective tissue. There is very little cicatricial residue in lungs with healed, benign, primary disease. Scar-tissue is only necessary for the cicatrization of ulceration, and ulceration may be prevented by the timely and proper use of potent antiallergics in combination with the antibiotics and chemical antibacterials now available.

Of course not until an ultra-potent antibiotic or chemical antibacterial, powerful enough for a quick "therapia sterilisans magna"

in tuberculosis is discovered, will both allergy and immunity cease to play their leading role in the disease.

Tuberculosis is not merely an infectious disease. Tuberculosis is really an infectious-allergic disease.

Already this suggested dual, antibacterial-antiallergic therapy, is being successfully applied to rheumatic disease, the other well known infectious-allergic disease.

The same may apply to other diseases that may have a basal, sensitizing, low grade infection in their substratum, but overshadowed by superimposed allergic manifestations, dominating their pathologic and clinical pictures. The type of disease in which ACTH and cortisone instead of acting as miraculous, curative agents, only act as miraculous palliatives, by their limited power to control, only, the superimposed, overshadowing allergic manifestations, but in which the combined use of ACTH or cortisone with simultaneous administration of the necessary antibiotic, or chemical antibacterial, for the simultaneous elimination of the basal, sensitizing, substratal infection, may result in the real, permanent, miraculous cures so anxiously awaited.

Maybe Rimifon has antiallergic beside antibacterial properties and is the ideal drug for dual antibacterial-antiallergic therapy.

FE DE ERRATA

En el trabajo del Dr. Luis M. Morales (Some Psychiatric Aspects of Sexual Impotence), publicado en nuestro número anterior, las primeras tres líneas en la página 350 deben decir así:

“1. Fear

- a. Of disapproval or punishment.
- b. Of criticism or ridicule.

CARCINOMA OF THE RECTUM COMPLICATING PREGNANCY

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Bayamón Charity District Hospital

Bayamón, P. R.

Carcinoma of the rectum complicating pregnancy is considered to be relatively rare. In an excellent review of the available literature on this subject made by Banner, Hunt and Dixon¹ from Mayo Clinic they encountered sixty two cases of carcinoma of the large bowel and added seven cases observed in that clinic in the past twenty seven years. The maternal mortality, which was recorded in forty one of the sixty two cases, was 63 per cent. The fetal mortality, which was recorded in forty of the sixty two cases, was 50 per cent. In their seven cases, the maternal mortality was 14 per cent, and the fetal mortality 28 per cent.

In 1952, Jennings² found in the literature seventy-seven cases of carcinoma of the large bowel complicating pregnancy and added another case.

In the last two years we have observed two cases of carcinoma of the rectum complicating pregnancy. Both of them were diagnosed during the third trimester of pregnancy. The report of these cases follows.

Case #1 - J. A. - Hospital #50925. A 37 years old G V P IV was admitted to the Fajardo District Hospital on 1/5/51 in the eighth month of pregnancy. Three years prior to admission she was treated for pulmonary tuberculosis with artificial pneumothorax, and later on thoracoplasty in the right side with good results. She gave a history of progressive constipation of about ten months duration with the passage of very thin caliber stools in the last three months. She was having frequent urge to defecate and passed small amounts of mucosanguinous material mixed with small amounts of feces, in the last few months. On physical examination she presented evidence of a right thoracoplasty without evidence of active pulmonary tuberculosis. The gravid uterus was of the expected size of an eight months pregnancy. In the rectum there was a stenosing lesion at two inches from the anal sphincter, hard, irregular and fixed to the perirectal tissues. Biopsy of this lesion was reported as adenocarcinoma. The laboratory studies showed a hemoglobin of 78% ; 4.0 millions red blood cells; 9,800 white blood cells with a normal differential count, urinalysis was

negative, the serologic test for syphilis was negative, and the Frei test was negative.

For reasons beyond our control intervention in this case was delayed up to 2/5/51 when a low cervical cesarean section was performed. A living baby weighing seven pounds was delivered. The patient had an uneventful postoperative course and this was followed four weeks later by an abdominoperineal resection (Mile's) which was tolerated well. Adhesions from previous surgery offered very little difficulty. Metastatic nodes in the area of the superior hemorrhoidal artery were felt. Histologic report on the resected sigmoid and rectum confirmed the diagnosis of papillary adenocarcinoma. (Fig. 1)

In 7/9/51 (approximately four months after the resection) she presented metastatic nodes in both groins confirmed by histologic study of nodes obtained at biopsy (Fig. 2). This was followed by evidence of spreading carcinomatosis in the abdomen with signs of partial bowel obstruction and death eight months after resection of the primary lesion.

Case #2 - G. W. M. - Hospital #98177. A 40 years old Negroess was admitted to the Bayamón District Hospital on 10/15/51 with the chief complaint of bloody bowel movements. Two years prior to the present pregnancy she passed bright red blood per rectum. It would come out at the beginning or end of defecation and was episodic. No pain or other associated symptoms. At two months pregnancy had recurrence of bleeding per rectum until seen on admission. Bowels moving regularly. No weight loss, in fact had gained weight during pregnancy.

She had had eleven previous pregnancies, nine of which terminated in normal spontaneous deliveries at term and two in early spontaneous abortions. The present pregnancy was of about 6-1/2 months duration.

The patient was found to be a well developed and well nourished Negro woman. The head, neck, eyes, ears, nose and throat revealed no evidence of disease. The breasts felt normal. The cardio-respiratory systems were within normal limits. Arterial blood pressure in the arms was 130/94. The examination of the abdomen revealed a gravid uterus with the fundus 3 cm. above the level of the umbilicus. The fetus was found in vertex presentation with the head in the right iliac fossa. Fetal heart rate was 136 per minute, in right flank, tone of good quality and regular. On pelvic examination the cervix was soft and clean, with no evidence of disease. The adnexae were normal. Rectally, a small, fungating mass was felt in the anterior wall, about 1-1/2 inches from the anal verge. There was no fixation or other evidence of a spreading neoplastic lesion.

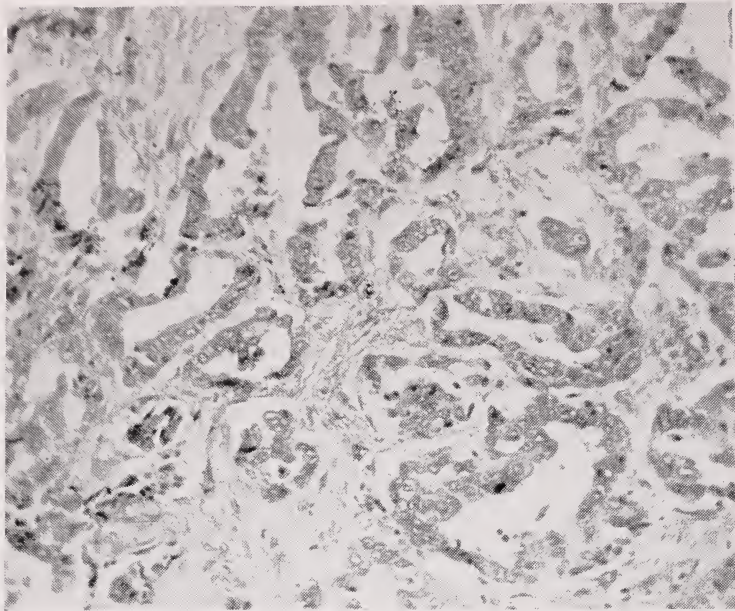


Fig. 1. Extensive tumoral infiltration of colonic wall by atypical glands lined by hyperchromatic columnar or cuboidal epithelium. (Mag. 230X)

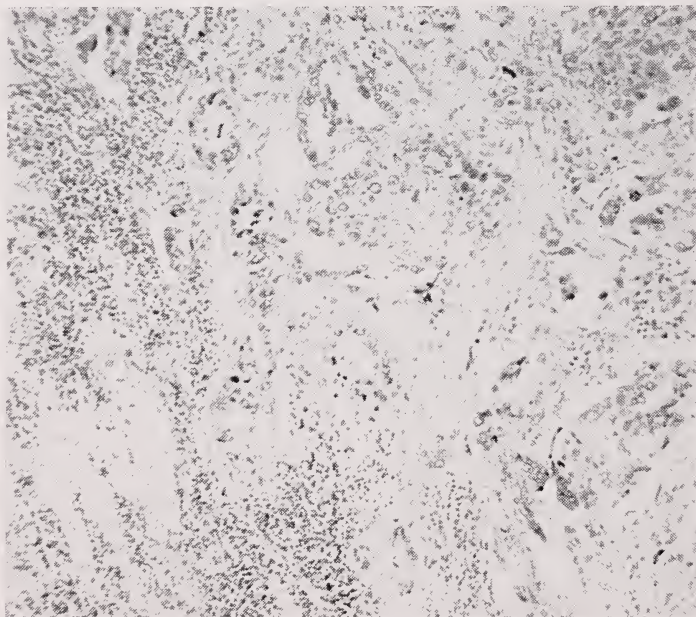


Fig. 2. One field of the metastatic carcinoma in the inguinal lymph glands. Note the similarity of the histologic patterns to that of the primary tumor. (Mag. 230X)

A biopsy of this lesion revealed a colloid carcinoma of the rectum. The red blood cell count was 3.39 millions with 9.4 gm. hemoglobin. The white blood cell count was 10,050 with a normal differential count, except for a 9 per cent eosinophilia. The urinalysis showed no abnormality. The serologic test of syphilis was negative. The bleeding and clotting times were normal.

A vaginal cesarean section with the aid of metreuryesis was performed on 10/29/51 (at the end of the seventh month of gestation). The baby was easily delivered, alive, by internal podalic version and extraction. The postoperative course was uneventful.

Comment: Delivery from below was chosen as the safest method. Since the pelvis was large and the child was small it was decided that a vaginal cesarean section was the procedure of choice. This procedure, even though rarely indicated, offered in this instance a safe and simple way to terminate the pregnancy. It, being an extraperitoneal section, lessened the danger of infection (peritonitis) and provided for a more suitable operative field to the surgeon a few weeks later by preventing the formation of adhesions in the pelvic cavity. Metreuryesis was used to make the cervix more accessible and to test the size of the opening in order to eliminate the danger of tears while doing the version and extraction.

After proper preparation for large bowel surgery an abdomino-perineal resection of the rectosigmoid with resection of the posterior vaginal wall was performed on 11/15/51, (16 days after terminating pregnancy). Spinal anesthesia was employed. The patient received 500 cc whole blood during the operation.

Operative findings: A tumor mass with a central ulceration was found in the anterior wall of the rectum and three centimeters from the mucocutaneous border of the anus. The ulcer measured two centimeters in its greatest diameter. The borders of the tumor mass were elevated and very hard. There was a direct extension of the tumor to the recto-vaginal septum. The vaginal mucosa, however, did not appear ulcerated. The liver was free of any metastatic lesions. There was no adenopathy of the mesosigmoid or periaortic region. The uterus was slightly increased in size and with the consistency of a normally involuting organ. The ovaries and Fallopian tubes appeared unremarkable. The cervix presented recent, healing anterior and posterior incisions but no evidence of ulceration or any metastatic lesion.

A permanent colostomy was done by bringing out the proximal end of the descending colon through a muscle splitting incision in the left lower quadrant of the abdomen. In the perineal dissection, the anus, rectum and adjacent pelvic floor musculature (levator ani) were excised en bloc, together with the posterior vaginal

wall. The vaginal wall was excised up to two centimeters from the cervix. The lateral walls of the vagina were then undermined and approximated with interrupted #0 chromic catgut so as to permit the introduction of two fingers into the newly reconstructed vagina. The vagina and the perineal opening were then packed with two-inch gauze strips.

PATHOLOGIC REPORT

Gross description: The specimen consists of a portion of the lower large bowel measuring 16 cms. in length and 6 cms. across its main diameter. It includes rectum, anus and perianal skin. In its anterior aspect the specimen contains a portion of the recto-vaginal septum including one area of 2.5 cms. of the vaginal wall. The serosa is smooth and glistening. The recto-vaginal septum is dense and very hard on palpation. The perirectal fat is grossly normal. No enlarged lymph nodes are seen on dissection. On opening the rectum, the mucosa is of a normal appearance in almost its total extension except in the area where the recto-vaginal septum is attached where it presents a rounded ulceration measuring 2 cms. in diameter. Its borders are elevated and irregular and are surrounded by congested and discolored edematous mucosa. Its base is occupied by a grayish granular or necrotic soft friable tissue. On sectioning the tissues are soft and granular in the ulcerated area. The walls of the rectum appear infiltrated by opaque granular whitish tissue which invades the perirectal structures just to the recto-vaginal septum. The vaginal wall is apparently free of tumoral invasion. The area of invasion of the tumor is circumscribed to one area approximately 0.5 cms. in the outermost limit at the ulcer. (Fig. 3).

Microscopic description: The mucosa, muscular layer and serosa of the rectum are extensively replaced by a tumoral growth made of variously sized, well differentiated tumoral glands lined by tall columnar cuboidal or flat epithelium. Many of them are dilated by mucoid secretion while others are tiny, showing a very narrow lumen. In areas the tumor shows a papillary pattern. No infiltration with isolated spheroidal or signet ring tumoral cells is seen. The recto-vaginal septum is infiltrated with isolated tumoral glands. (Fig. 4).

Diagnosis: Ulcerating colloid carcinoma of rectum with extension to the recto-vaginal septum.

The postoperative course was entirely uneventful. The colostomy functioned well. The incision healed by primary intention.

In the last follow-up visit (3/21/52) her general condition was good. The incisions are healed. The colostomy has continued

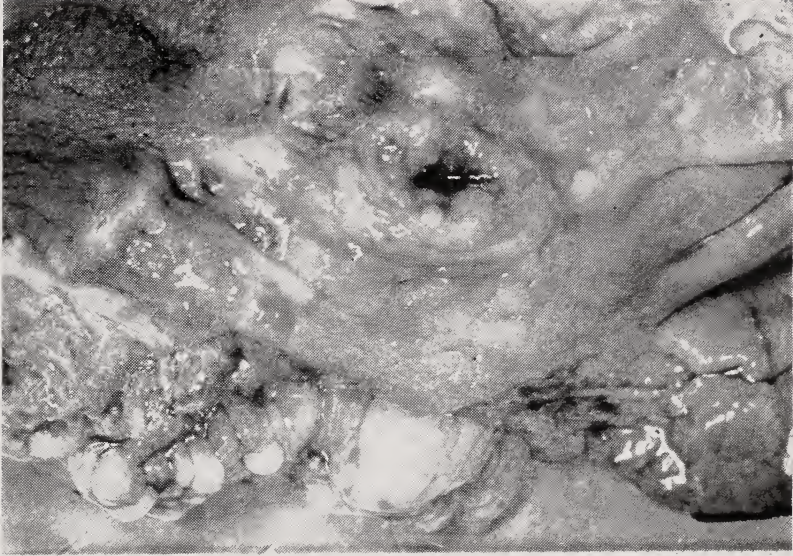


Fig. 3. Gross photography of the ulcerative carcinoma of rectum diagnosed by previous biopsy. Note the central crateriform ulceration of the tumor. The rectovaginal septum was invaded by the carcinoma (about 2/3 natural size).



Fig. 4. Tumoral glands are infiltrating the muscular layer of the colon. The tumoral epithelium is differentiated. Most of the glands are undergoing colloid degeneration. (Mag. 100X)

functioning well. The vagina admits two fingers and has five inches of depth. The patient has gained six pounds in weight since she was discharged from the hospital. There is no evidence of local recurrence or generalized metastases.

DISCUSSION

The symptoms produced by the neoplastic process in the rectum can be mistakenly ascribed to the pregnancy. Constipation and rectal bleeding were the presenting symptoms in our cases. At times the first manifestation of this lesion has been acute intestinal obstruction.^{5, 1} Other symptoms of this disease are irregular attacks of diarrhea, and occasional abdominal crampy pain. Usually there is no weight loss possibly because of the increasing bulk of the pregnant uterus. The mild secondary anemia may be attributed to the physiologic anemia of pregnancy. A moderate increase in the sedimentation rate of the erythrocytes accompanies pregnancy as well as carcinoma.⁴

Constipation that does not respond to the usual measures employed, after a reasonable period of time during pregnancy, demands a thorough study of the large bowel. Bleeding per rectum although frequently is secondary to hemorrhoids, developing or aggravated during pregnancy, should be investigated and especially if accompanied by some of the symptoms mentioned above.

In our cases the diagnosis was established by digital examination of the rectum. This was confirmed by histologic examination of the tissue obtained by biopsy of the lesion through the proctoscope. The early diagnosis of carcinoma of the rectum is most important if the mother is to be salvaged.

The management of this problem is best approached by evaluating the degree of operability of the carcinoma and estimating the stage of the pregnancy. The first factor can not be established before opening the abdomen except when there is obvious metastatic involvement of other organs. The tendency at present is to treat the carcinoma independently of the pregnancy. Bacon,² quoting Maud Slye, states that extragenital cancer (except in the breast) is not influenced by pregnancy, and, therefore, if this is true there is absolutely no indication for hysterectomy or abortion. Bainbridge, quoted by Jennings,³ feels that pregnancy speeds up all cancer growth.

Each case should be treated individually, but certain general principles employed in the cases reported by others can be followed. In the first and second trimesters, the lesions should be resected either by abdomino-perineal resection or by anterior resection with end-to-end anastomosis regardless of the pregnancy.

If the uterus is involved by the malignant lesion, then it should be included in the resection. In the third trimester, cesarean section should be performed in the seventh and eighth months. Resection of the malignant lesion should be made at this time or after two to four weeks, depending on the status of the patient. In the ninth month labor may be induced with delivery from below if the lesion is not too large, followed in two to four weeks by resection, or the criteria set down for the seventh and eighth months may be used. If an abdomino-perineal resection is done early in the pregnancy, the delivery of choice should be from below, unless there is some contraindication on pelvic examination.

SUMMARY

Two cases of carcinoma of the rectum complicating pregnancy are presented, elevating to 80 the number reported in the literature. The management of this problem is discussed.

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GUILLAIN-BARRE SYNDROME

CASE REPORT

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In 1916 Georges Guillain, J. A. Barre and A. Strohl reported two cases of French soldiers suffering from flacid paralysis of their lower extremities, abolition of tendon reflexes, preservation of cutaneous sensations and acellular hyperalbuminosis of the spinal fluid. This syndrome was called radiculoneuritis with acellular hyperalbuminosis by these investigators. Since then, several other cases have been reported by other workers and have been called acute polyneuritis with facial diplegia, acute febrile polyneuritis, myeloradiculitis, infectious polyneuritis, etc. All these clinical forms represent the same disease and are more commonly called Guillain-Barre Syndrome after the original investigators.

Etiology: The cause of the disease is unknown. In 1918 Bradford, Bashford and Wilson, injected an emulsion of a spinal chord of a fatal case into the subdural space of a monkey and the disease was reproduced in the animal. It was then carried from monkey to monkey in series. They then succeeded in isolating globoid bodies in culture on Noguchi's media. These globoid bodies were similar to the ones isolated by Flexner and Noguchi in acute poliomyelitis, and because of this evidence they concluded that a neurotropic virus was the cause of the disease.

Later work failed to reproduce these results, and in 1941 Sabin and Aring made emulsions of the brain and spinal column of three fatal cases and injected mice, guinea pigs, and monkeys without being able to reproduce the disease. At the time of this work these investigators were trying to isolate the virus which causes pleuro-pneumonia, which was blamed for Guillain-Barre's Syndrome by other workers. No virus or organism could be isolated by Aring and Sabin and they finally expressed the conclusion that the disease was probably produced by the toxins of the microorganisms responsible for the infection of the respiratory tract which usually preceded the onset of nervous symptoms.

A syndrome similar to Guillain-Barre's has been described in troops serving in the tropics after faucial and cutaneous diphtheria, so it might be that infectious polyneuritis is due to several noxious agents which give rise to an allergic reaction producing this syndrome.

Pathology: In 1918, Bradford, Bashford and Wilson first described the pathological picture which they thought was limited to

the peripheral nerves and the posterior root cells. These changes were: 1) marked edema of nerve bundles, 2) congestion, 3) moderate increase in cellularity with a tendency to focal accumulation of cells, 4) swelling and beading of the myelin sheath and swelling, beading and fragmentation and dissolution of the axis cylinder.

Since then it has been found that not only the peripheral nerves and central nervous system is involved, but that the adrenal glands, the liver, the kidneys and the heart show pathological changes.

Sabing and Aring, in 1941, in a series of three postmortem examinations reported glial proliferation of the posterior root cells, degeneration of the posterior root cells, generation of the anterior horn cells, and degeneration of the facial and trigeminal motor nuclei at the pons. The cerebrum was reported to show edema and infiltration of glial cells in the cortex. The viscera, as stated before, were also involved. The adrenal glands show three distinct type of lesions: 1) foci of degenerated cortical cells, 2) infiltration with mononuclear cells along the adrenal gland nerves, 3) focal accumulation of lymphocytes and plasma cells occupying what seemed to be lymphatics spaces or degenerated cortical cells.

The hearts examined at postmortum showed: 1) diffuse interstitial infiltration with mononuclear and polymorphonuclear cells, 2) areas which suggested necrosis of isolated muscle fibers, infiltrated by phagocytic cells, 3) focal phlebitis affecting the coronary veins.

The liver showed: 1) focal necrosis involving a few cells with a local infiltration of mononuclear and polymorphonuclear cells, 2) interlobular infiltration of connective tissue or portal canals with mononuclear and polymorphonuclear cells.

The kidney in two of the three cases studied showed extensive interstitial infiltration with mononuclear cells specially in between the tubules. The adjacent vessels were congested but the tubules and the glomeruli were intact, except for some occasional glomerulus which showed fibrosis and obliteration. These changes were similar to the ones described by Councilman in certain patient suffering from scarlet fever and diphtheria.

In all three cases there was some lung involvement. In all three cases there was acute bronchitis and lobular pneumonia present and in one case there was a mild, focal, acute colitis. In none of the sections was there any evidence of inclusion bodies, although special care was taken in searching for them.

Age and sex: The disease is more common in young adult or in early middle life. In 26 cases reported by F. Foster, M. Brown and H. H. Merrit in 1941, the average age was 35 years. The youngest patient was 2 years of age, and the oldest was 78 years

old. It seems to be more common in males with a ratio of 4:1, but the prognosis is worse in females.

Symptomatology: The onset is preceded by an acute respiratory infection in about 50% of the cases. Following the respiratory infection there is a latent period that may last from a few days to several weeks.

After this preceding infection the patient feels well and then develops muscular pains which usually begin in their lower extremities and lumbar region. Throughout the course of the disease the temperature usually remains normal but the pulse rate is usually elevated with an average of about 95 beats per minute. Muscle and nerve tenderness is usually present but might be absent. Weakness of the hands and feet may develop suddenly or might take days after the onset of the pain. The weakness progresses into a bilateral ascending flaccid paralysis, starting in the distal portion of extremities and then involving the complete limb usually bilaterally. At times the thoracic and abdominal muscles are involved. Despite the severe flaccid paralysis, muscle atrophy is rare. Muscle fasciculation, signs of anterior horn cell disease are usually absent, but they may be present. Sphincter's involvement is rare.

The tendon reflexes are absent in all the affected muscles and in some instances are absent in uninvolved muscle groups. The abdominal and cremasteric reflexes are usually absent. The Babinski and Hoffman reflexes are always negative.

In 85% of the cases there is cranial nerve involvement and the most commonly affected nerve is the facial nerve. This involvement is usually bilateral causing facial diplegia. Other cranial nerves may be involved producing oculomotor paralysis, weakness of the masseters, palatal weakness, etc. The phrenic nerve usually escapes.

In 50% of the cases reported by Foster, Brown, and Meritt there was sufficient respiratory embarrassment to need the use of a respirator and they reported a mortality rate of 42% in their series of 26 cases.

Laboratory Data: The white cells count varies from 6,200 to 22,000, with an average of 10,000. The red blood count varies from 3,000,000 to 6,000,000 with a hemoglobin range of 58% to 108%.

The urine is usually negative, although in some cases albumin and WBC are present.

The cerebrospinal fluid is diagnostic in this disease. There is a hyperalbuminosis with no increase in the cell count. The average spinal fluid protein is about 200 mgm%. The rest of the chemistry is normal. There are no specific changes in the spinal fluid chlorides or sugar. The Wassermann test is negative. The

colloidal gold test may show a first or a middle zone curve. The spinal fluid pressure may or may not be elevated. A theory has been promulgated to explain this increase in protein in the spinal fluid. Some investigators have stated that the spinal fluid in the spinal column is normally drained around the spaces which surround the dorsal ganglia of the spinal column, and in this disease the edema of the myelin sheath impairs the absorption of proteins by the general circulation.

Differential Diagnoses: Guillain-Barre's Syndrome has to be differentiated from neuritis due to lead poisoning or to vitamin B deficiency, anterior poliomyelitis, syphilis, Landry's ascending myelitis, myxedema, diabetic neuritis and spinal chord tumor.

The polyneuritis due to lead intoxication can be differentiated by the history, the peripheral blood picture, the lead deposits on the bones, and the lead levels of the urine and blood. The paralysis is usually motor and nerve pain is not a common finding. The spinal fluid is normal.

Polyneuritis due to vitamin B deficiency is characterized by its mildness, paresis is more frequent than paralysis. At times there is involvement of the spinal column producing ataxia, loss of position and vibratory sense. These senses are preserved in Guillain-Barre's Syndrome. The spinal fluid findings would also differentiate these conditions.

Syphilis of the central nervous system is easily differentiated by the spinal fluid and peripheral blood serology.

Anterior poliomyelitis is separated by the history of fever, the meningeal signs, the presence of cells in the spinal fluid and the more rapid development.

Myxedema and diabetic neuritis can be confused because in both conditions there may be an increase in protein in spinal fluid without an increase in cells, but the clinical picture in myxedema is different, and the blood sugar elevation in diabetic neuritis will immediately clear up the diagnosis.

In Landry's ascending paralysis the diagnosis might be more difficult because in both conditions there is an ascending paralysis, but they might be differentiated clinically because of the lack of muscle and nerve tenderness in Landry's and because of the spinal fluid which is essentially negative in Landry's.

Spinal chord tumor would give localizing signs and the spinal fluid hydrodynamic would serve to differentiate these two conditions.

Prognosis: Guillain and Barre considered their syndrome as very benign with a universal complete recovery, and in 1936 they again stated that they have had complete recovery in all their cases (14 cases). Since then other reports have described fatalities

in some series, even up to 40%. (Brown, Merrit and Foster, 1941).

In 1942, Fox and O'Connor made a review of the cases reported in the literature up to that time and found that 26 out of 126 cases had ended fatally: a mortality rate of 20.6%.

Treatment: There is no specific treatment; prostigmin sulfate, B.A.L., and X-ray therapy have been tried with indifferent results.

Sidney Stillman and Gonesy recently treated a case with ACTH and Cortisone with gratifying results.

CASE REPORT

Name: M. R. P. - Pfc.

Past Medical History and Family History: Essentially negative, except for a head injury with skull fracture at the age of ten.

History of Present Illness: M. R. P. is a 27-year-old male that was admitted to Rodríguez Army Hospital on 18 June 1951, complaining of extreme weakness of the lower extremities, inability to open his jaw, pain on his calf muscles bilaterally, pain on his lumbar region, and numbness of hands and feet. The patient was well until 6 June, 1951 when he developed an acute respiratory infection which was followed two days later by numbness of his hands and feet. On 10 June 1951 he noticed weakness and numbness of his arms and forearms, and a day later he developed weakness and extreme tenderness of the calf muscles. The patient was transferred from Camp Salinas to Camp Losey, for medical treatment. In this installation he was treated with salicylate and bed rest with no improvement. On 13 June 1951 he noticed weakness of his jaw muscles and difficulty in speaking. On 14 June 1951 he collapsed while coming back from the toilet. At that time he had extreme weakness of his lower extremities and tenderness on the lumbar region. Because of these complaints he was finally referred to Rodríguez Army Hospital for evaluation, treatment, and final disposition, on 18 June 1951.

Physical Examination on Admission: There were no signs of injury on palpation and examination of his skull. Eyes were normal to light and accommodation. The oculomotor nerve was intact. There was extreme tenderness on palpation of the masseter muscles. Lungs were clear to auscultation and percussion. Blood pressure was 180/100. Pulse was 90. Heart had a normal rhythm and no murmur. The abdomen was essentially negative. The abdominal reflexes were present but depressed.

Neurological Examination: The cranial nerves were intact at the time of admission, except for slight weakness of the motor nucleus of the trigeminal nerve. There were diminished biceps and

triceps reflexes on the upper extremities. There were absent Achilles and patellar reflexes. There was no neck rigidity, and no abnormal pathological reflexes present. There were some areas of paresthesia on the hands and feet. Position and vibratory senses were intact. The abdominal and cremasteric reflexes were diminished but present. Tenderness was very severe on the gastrocnemius and masseters muscles.

Laboratory Examinations: Blood count on 19 June 1951 was reported as follows: RBC - 5,420,000; 16.5 Hb; WBC - 11,450; with a differential count of 55% neutrophils; 39% lymphocytes; 5% eosinophils, and 1% stabs with no atypical lymphocytes. Serology and urinalysis were negative. Spinal fluid revealed no cells. Total protein was 249 mgm% and 90 mgm% of sugar, and a smear taken for organism was negative. The spinal fluid Wassermann was also negative. Sedimentation rate on 19 June 1951 was 5 mm per hour. A spinal fluid culture was also reported negative. Another spinal fluid examined on 19 June 1951 gave the following findings: No cells were seen. Sugar was 72 mgm%, total protein 322 mgm%, and Chlorides, 620 mgm%. Another stained smear examined revealed no organisms. Throat cultures on 20 June and on 19 June 1951 were negative for *C. diphtheria*. Repeated blood counts on 25 June 1951 revealed: RBC - 5,670,000, with 19% Hb; WBC - 14,400; 71% neutrophils, 27% lymphocytes and 2% eosinophils. Blood chlorides determination on 26 June 1951 were 364 mgm%. A repeated spinal fluid on 2 July 1951 revealed 192 mgm% total protein, with no cells. Blood urea nitrogen on 3 July 1951 showed 15 mgm%. Serum proteins on 6 July 1951 gave the following results: total proteins - 7.44 gm%, albumin - 4.40 mg%; globulin 3.04 mgm%, an A/G ratio - of 1.1:1. Cephalin flocculation was 3+, thymol turbidity, 6 units.

Course in Hospital: On admission the patient was acutely ill and showed paralysis of his lower extremities and of his masseters muscles. At 20:00 hours on the day of admission the man had two convulsions which were treated with sedation; next morning the patient showed weakness of masseters, fasciculations of his pectoralis muscles and a tachycardia of 130 which was interpreted as involvement of dorsal nucleus of his vagus. At 08:00 hours he had another severe convulsion and a spinal tap was done to relieve his increased cerebrospinal fluid pressure.

In spite of this evidence of cerebral edema and bulbar involvement the man never developed paralysis of his phrenic or intercostal nerves.

From the second hospital day on, the patient improved slowly until he recovered completely, except for the absence of his tendon reflexes which persisted absent on discharge.

It is interesting to observe that this man showed an A/G ration of 1.1:1, a cephalin flocculation of 3+ and thymol turbidity of 6 units and liver tenderness as evidences of liver involvement. No heterophile antibodies were obtained but atypical lymphocytes were persistently absent in his peripheral blood.

X-ray of heart and electrocardiogram failed to show any evidence of cardiac involvement so it was assumed that his prolonged tachycardia was due to involvement of his motor vagi. With improvement of his neurological symptoms his cardiac rhythm as well as his blood pressure returned to normal.

This is a case of Guillain-Barre's Syndrome showing cerebral involvement characterized clinically by convulsions, anterior horn involvement as evidenced by muscular fasciculations, bulbar involvement with tachycardia of 130 and liver impairment with a cephalin flocculation test of 3+, thymol turbidity of 6 units and an A/G ratio 1.1:1.

Resume: A case of Guillain Barre's showing evidences of cerebral, bulbar, spinal column and liver involvement is reported.

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CINCUENTA AÑOS DE MEDICINA ORGANIZADA*

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Fué en diciembre de 1947 y durante la Cuadragésima-cuarta asamblea de esta Asociación, que tuve la oportunidad de hacer un raquítico recuento, de lo que la medicina nativa había hecho en pro de la regeneración social y física del pueblo de Puerto Rico, durante la etapa comprendida entre los años 1850 y 1900.

En esta ocasión histórica, al cumplirse el primer cincuentenario de la fundación de la Asociación Médica de Puerto Rico, es otra vez mi privilegio, aunque en cinemática exposición, hacer un balance de los aportes humanos, culturales y científicos, con que la medicina puertorriqueña marcó su ritmo durante la etapa del año 1900 al año 1950.

Decíamos entonces, que la medicina y las exigencias intelectuales de los médicos de la época habiendo establecido como triunfo inicial el tráfico del libro, dedicaron el esfuerzo generoso de su influencia a promover la obra valedera de despertar la conciencia del criollo.

Fueron las mismas corrientes intelectuales que habían hecho realidad la revolución francesa y la liberación de las colonias americanas, las que infiltrándose a través del liderato médico representado por Betances y Padilla, por Alonso y por Corchado, hicieron viables los románticos episodios de Lares y de Yauco.

Cuando el 25 de julio de 1898 se asomaron por los ventanales de Guánica las tropas de Miles, y en histórica proclama ofrecieron las inmunidades y bendiciones de las instituciones liberales americanas, encontraron un liderato cívico cuya noble misión además de curar al enfermo, hacía armas nobles y valientes en las lides del periodismo militante; señalaba derroteros y aunaba voluntades en la doctrinación de la cátedra, y defendía aún a despecho de la idiotez enagenadora de nuestras masas campesinas, la tierra santa que codiciaban los inversionistas de la época.

Fueron entre otros muchos, Ferrer Hernández, del Valle Rodríguez, Coll y Toste, Zeno Gandía, Guzmán Rodríguez, Barbosa Brioso, Quevedo Báez, Font y Guillot y Felipe Cordero, la pléyade romántica de médicos puertorriqueños que, como sus predecesores en la gesta del Siglo XIX, iniciaron la nueva cruzada, que comenzaba bajo una nueva soberanía política, en los albores del Siglo XX. Asomémonos reverentes al ventanal de la época y observemos la jornada que con perfiles de tragedia, iniciaba el ritmo de la nueva vida.

* Discurso pronunciado durante el acto conmemorativo del cincuentenario de la Asociación Médica de Puerto Rico, 21 de septiembre de 1952.

Los médicos de pueblos y ciudades de Puerto Rico rendían sus servicios profesionales a la comunidad, vistiendo el humilde uniforme del médico titular o la clámide pomposa del facultativo particular. Muchos de ellos, especialmente en las comunidades pobres y pequeñas, ejercían el doble ministerio, pero el desideratum de la prestancia era ser médico particular.

Ejercer como médico titular en una población, conllevaba subvención raquítica pagada a destiempo; horas de trabajo fuera de toda norma razonable; riñas con los alcaldes, botas enlodadas y cabalgadura a la puerta. Por el contrario, el médico particular también gozaba de honorarios raquíuticos pagados de cosecha en cosecha; horas de consulta señaladas casi siempre al terminar la hora amable de la siesta; terno de dril blanco inmaculado y caleza con faroles de carburo.

En las grandes urbes la diferencia era más notable. Los médicos particulares visitaban sus enfermos en cómodos faetones, pero muy frecuentemente hipotecaban la casa solariega, para sufragar los gastos de viaje a Madrid o a Nueva York.

Los médicos titulares mientras tanto, caminaban jadeantes por la vuelta del Diablo, por las callejuelas del barrio de Balboa, o por los enmarañados callejones del Pastillo.

El espectáculo que presentaban novecientos cincuentitrés mil doscientos cuarentitrés puertorriqueños laborando con desesperación y con desnudo en una Isla de 3,435 millas cuadradas, mil de ellas solamente dedicadas a labrantío, era de desolación y de miseria. Si a ese balance de tétricas proyecciones se agrupan los relieves de una agricultura cafetera arruinada en su totalidad por el temporal de San Ciriaco, y una industria azucarera empobrecida pero altamente codiciada por los inversionistas de Louisiana y Nueva York, el saldo no puede ser más angustioso y sus proyecciones económicas más detrimentales.

El Congreso de los Estados Unidos ante el desastre horrendo del huracán de agosto 8, 1899, apropió \$200,000 para socorrer los millares de víctimas que quedaron al margen de la caridad pública, cantidad que resultó totalmente inadecuada ante la magnitud del problema.

Las masas campesinas que habían sido calificadas por un médico puertorriqueño de aquella época, el Dr. Gonzalo Córdova, como las más anemiadas del universo, remedaban en los días que siguieron al devastador huracán, legiones espectrales que imploraban limosna a las orillas de los caminos.

En medio de ese cuadro, el 24 de noviembre de 1899, el Capitán Médico Bailey K. Ashford, destacado en Ponce, telegrafió al Cirujano General en San Juan, que había comprobado que muchas de las anemias progresivas de la Isla se debían al ankylostoma duo-

denal. Esta revelación científica que tuvo Ashford en una de las etapas más tristes del campesino puertorriqueño, tuvo una vital trascendencia en la rehabilitación de la inmensa mayoría de nuestras masas campesinas de entonces.

Tres años más tarde, el 21 de septiembre de 1902, el Dr. Manuel Quevedo Báez, en unión de una decena de médicos fundó la Asociación Médica de Puerto Rico. Alrededor de esa primera gesta de la medicina organizada, además, de la figura prócer del eximio fundador, los nombres de Vélez López, Saldaña, Pepe Carbonell, Narciso Dobal y Mariano Ramírez entre otros, merecen la ofrenda de nuestro recuerdo y nuestra gratitud.

En enero de 1903, bajo la dirección del Dr. Ramón Ruiz Arnau, el primer número del Boletín de la Asociación Médica Puertorriqueña vió la luz pública, y con esta publicación se vincularon los primeros esfuerzos de la producción científica de la clase médica. Fueron los Dres. Goenaga, Coll y Toste, Ruiz Arnau, Pedro del Valle y Quevedo Báez los primeros en romper lanzas desde las columnas del Boletín, y fué desde este reducto donde se iniciaron las primeras intentonas revolucionarias de legislación médica, entre ellas la creación del Tribunal Examinador de Médicos.

En ese mismo año — septiembre de 1903 — Pedro Gutiérrez Igaravidez, que más tarde había de ser un fervoroso colaborador de Ashford en la campaña antiuncinariásica de Puerto Rico trajo ante la Sección de Estudios Patológicos de la Asociación Médica, las primeras demostraciones micrográficas del plasmodio de Laveran, iniciando de ese modo el estudio científico de nuestras fiebres, y aclarando de esa manera el campo más oscuro de nuestra medicina regional.

Fueron aquellos primeros días de urgente reorganización, bajo un nuevo régimen, severísimas pruebas para el conglomerado puertorriqueño pero especialmente para su clase médica. En enero de 1903, cuando ya la Asociación contaba entre sus socios, un centenar de prestigiosos médicos, el presupuesto mensual de la misma era de \$75.00, y en la asamblea anual de diciembre del mismo año, se discutió ardorosamente la conveniencia de rebajar la cuota mensual, que era de \$1.00. Es posible que los \$83.33 — la celeberrima paga de esa etapa — no permitiese a la mayoría de los médicos sufragar las cuotas de la naciente asociación.

La cuota anual de \$12 se pagaba en semianualidades vencidas, y según parece con frecuentes y perjudiciales dilaciones. Esas frecuentes dilaciones obligaban a la tesorería de la Asociación, a verificar cobros a domicilio para poder cumplir las erogaciones del humilde y raquítico presupuesto.

La clase médica puertorriqueña en su inmensa mayoría, oficiaba en el altar de la más extremada pobreza. Evoca mi memo-

ria en el arqueo de los recuerdos de entonces la figura marchitada por los años y dolencias físicas, de aquel pobre viejecito, gangoso y derrengado, que era en nuestras oficinas de la Calle O'Donnell, Secretario Ejecutivo, Portero, Conserje y Cobrador. Este celoso cancerbero de la maltrecha Asociación, visitaba anualmente las oficinas de los asociados de la Isla, implorando el pago de las cuotas vencidas y atrasadas.

En medio de todas esas tribulaciones, el Dr. Isaac González Martínez, a mediados del 1904, anunció a la profesión médica y al pueblo de Puerto Rico, el descubrimiento de la bilharziosis en el hijo del país, encauzando con los descubrimientos de Ashford y Gutiérrez Igaravidez, los primeros logros de las investigaciones médicas en la lucha tenaz por la salud de nuestro pueblo que de manera tan gallarda se iniciaba.

En el 1909, era Presidente de la Asociación Médica de Puerto Rico, el sabio médico aguadillano doctor Agustín Stahl. Ocupaba la presidencia desde el 1905 y en junio de 1909 apareció en La Correspondencia de Puerto Rico, un comunicado bajo su firma, que decía como sigue:

“Aviso a los señores médicos de la Isla. Por la presente les hago saber por lo que pueda interesar, que me he desligado en absoluto de la Asociación Médica de Puerto Rico, de la que he sido Presidente desde enero de 1905 hasta ahora, obedeciendo a la indiferencia completa de los asociados observada desde hace un año, su retraimiento de este centro profesional y científico, primero y único creado en Puerto Rico por puertorriqueños, y otras razones de parecido carácter, por las que también conceptúo extinguida la Asociación.”

Llama extraordinariamente la atención, a despecho del tiempo transcurrido, que el violento comunicado del Dr. Stahl no fuese comentado por sus compañeros de Directiva los Dres. Gutiérrez Igaravidez, Vélez López y Carbonell.

El Boletín de la Asociación Médica de Puerto Rico tampoco vió la luz pública durante todo el año 1909 y gran parte del 1910, siendo tal vez justificado el epíteto de extinguida, que usara Stahl en sus vitriólicas declaraciones en La Correspondencia de Puerto Rico.

El 13 de noviembre de 1909, siete meses después del comunicado que declaraba extinguida la Asociación Médica de Puerto Rico, los Dres. Salazar, Suárez, Villaronga, Ruth y Salicrup de Ponce, invitaron a los médicos de la Isla a fundar la Asociación Médica de Puerto Rico, la cual según los prestigiosos firmantes de aquella convocatoria, estaba disuelta de hecho.

El 20 de noviembre del mismo año, siete días después de la convocatoria del grupo de Ponce, Pedro Gutiérrez Igaravidez, Rafael Vélez López y José V. Carbonel, citaron para una asamblea

general que se verificó en San Juan de Puerto Rico el 5 de diciembre.

En aquella histórica asamblea se renovó la junta de gobierno; se reformó el reglamento y se discutieron ampliamente las ventajas de la incorporación a la Asociación Médica Americana, que venía siendo defendida desde el 1907 por el Dr. Luis García de Quevedo.

Verificada la reforma, dos años más tarde en el 1911, se aprobó la ley organizando el Servicio de Sanidad Insular, y un año más tarde en el 1912 la Asociación contaba con ciento cincuenta y dos asociados y \$2,711.48 en sus arcas.

En el 1915 se fundó la Academia de Medicina de Puerto Rico. Los Dres. Ruiz Arnau, González Martínez, Gutiérrez Igaravidez, Ashford y Font y Guillot entre otros, fueron los líderes de aquel movimiento científico que tuvo entre sus logros, influenciar de una manera innovadora la producción médica literaria del país.

En el 1922, veinte años después de haber sido fundada la Asociación Médica de Puerto Rico, un millón setecientos veintitrés mil quinientos treinticuatro (1,723,534) puertorriqueños pululaban por las 3,435 millas cuadradas de la Isla. De esa cifra un millón, ciento cincuentiocho mil, novecientos dieciocho habitantes residían en la zona rural. El ingreso semanal de esa inmensa legión de jornaleros ascendía a la irrisoria suma de \$3.00, mientras el costo de alimentación alcanzaba a un promedio de \$3.19.

Es muy posible, que alrededor de un millón de puertorriqueños en esa etapa de nuestra vida, no pudiera pagar honorarios profesionales de índole alguna. Y es muy probable también que, de los setecientos veintitrés mil, quinientos treinticuatro restantes, un número considerable perteneciese a la legión inmensa que sólo percibía \$3.00 semanales, y como era razonable estuviesen inscritos en los empadronamientos municipales como pobres insolventes.

Muchos de los médicos aquí presentes no ignoran, por no haberlo podido olvidar, el número considerable de profesionales de ciudades y pueblos, que muy a pesar de la vida modesta y llena de privaciones que eligieron, no alcanzaban por virtud de la reinante crisis económica a pagar contribuciones por ingresos.

Desde esta misma tribuna se afirmó en 1947 y durante nuestra cuadragésima-cuarta asamblea, que a partir del 1915, el médico puertorriqueño había perdido el liderato cívico de su pueblo, habiéndose limitado el exponente a preguntar: ¿Por qué?

Aquella pregunta fué comentada por el entonces gobernador de Puerto Rico Don Jesús T. Piñero y por el atildado editorialista de El Mundo, Sr. Rivera Otero. Como era natural la medicina organizada, fué objeto de críticas injustificadas, que envolvían responsabilidades a que nunca fué acreedora.

Más tarde, en el 1949 y desde el Boletín de la Asociación Médica se intentó contestar la pregunta aduciendo el argumento de que tan pronto el médico puertorriqueño se hubo enfrascado en las múltiples investigaciones de la ciencia no había podido retener el liderato cívico, que los jefes políticos respetaron y temieron.

Es lástima que el romance de las investigaciones no fuese la causa real y efectiva. El pesado fardo de aquel liderato angustioso que la clase médica comenzó a sacudir en el 1915, y que estuvo basado principalmente en servicios médicos gratuitos que alcanzaban a grupos poblacionales de relativa prosperidad, era todavía angustioso impedimento en el 1935.

En aquel tráfico del respeto y del temor, la figura del médico disfrazado de líder, en no pocas ocasiones demandaba y obtenía el debido respeto que ofrendaba el temor, mientras esclavizaba su tiempo o su hacienda en holocausto de un liderato con alcances de dolor y de miseria.

En diciembre de 1934, a los veinte y cinco años de aquel episodio que en 1909, amenazara la vida de la Asociación, volvió ésta a sentir el impacto de la más cruel incomprensión en maridaje con la más cruel miseria colectiva. En esta ocasión, la clase médica puertorriqueña que apenas había podido sostener con decoro y con holgura la convivencia de una asociación, y que había luchado encarecidamente por que se rebajase la módica cuota de \$1.00 a setenta y cinco centavos, desmembró sus huestes bajo dos banderas, que intentaron reconocimiento y legitimación a través de las más burdas e infantiles especulaciones.

Aún viven y están presentes aquí esta noche, muchos de los actores principales de aquel drama, y a pesar de que en aquel entonces nadie enfocara el malestar reinante en proyecciones de latente crisis económica, muchos de ellos en el sosegado recuento de los acontecimientos de diciembre de 1934, creen que los factores fueron si no idénticos muy parecidos a los del 1909.

En agosto de 1936, el Dr. Manuel Pavía Fernández, abandona la presidencia de la Asociación Médica de Puerto Rico, con gesto lleno de abnegación y de hidalguía, en plan de reasociar las huestes en desacuerdo.

Al terminar la honda y larga crisis que mantuvo en honda disidencia a la Asociación Médica de Puerto Rico, el Dr. Juan Higinio Font fué exaltado a la presidencia el 12 de septiembre de 1936.

La creación de una Escuela de Medicina que ameritase el reconocimiento del Consejo de Educación y Hospitales de la Asociación Médica Americana, fué objeto del más absoluto respaldo en el 1942, y en aquel entonces se solicitó la creación de una comisión conjunta de la Legislatura y de la Asociación Médica de Puerto Rico.

En abril de 1944 la Cámara de Delegados de la Asociación Médica se expresó a favor del establecimiento en Puerto Rico de una Escuela de Medicina, al darse cuenta de un informe preliminar que había preparado para el Rector de la Universidad una comisión integrada por los doctores Costa Mandry, Suárez y de la Pila Iglesias.

En marzo de 1948 el Gobernador de Puerto Rico, don Jesús T. Piñero, reveló tener en su poder la oferta singular de la Fundación Paderesky, que ofrecía trasladar a nuestra Isla la escuela que tenía dicha fundación en Edinburgo.

No hemos de ahondar innecesariamente en la lamentable jornada de aquella escuela polaca que había sido forjada al calor de las necesidades imperiosas de la guerra y que su precaria subsistencia en los anales de la paz, hubo de convertirla, para desgracia nuestra, en la aparente canalización de nuestros más arraigados anhelos alrededor de la inmediata estructuración de nuestra escuela de medicina.

El Pueblo de Puerto Rico, la Escuela de Medicina de la Universidad de Puerto Rico y la clase médica puertorriqueña, tienen una deuda de gratitud imperecedera con el Dr. Manuel Angel Astor. Fué Astor, durante su presidencia de la Asociación Médica de Puerto Rico, quien con sobresalientes dotes de altivez y de energía logró librar a la Universidad y a Puerto Rico, de la más grande calamidad que amenazara el prestigio de sus instituciones educativas.

El más sosegado y minucioso recuento de las actividades cívicas y científicas de la Asociación Médica de Puerto Rico durante los últimos cincuenta años, necesariamente tiene que estar plagado de grandes injustas omisiones.

En el arqueo de las múltiples jornadas que atesora la historia de la clase médica puertorriqueña en este medio siglo de la vida ciudadana, las figuras militantes de los últimos decenios, habrán de ser aquilatados por las generaciones del futuro.

Hay sin embargo figuras humildes, muchas de ellas desaparecidas del escenario de la vida que soslayando arrogantes las crueldades de las omisiones y el olvido, demandan con justicia las siemprevivas del recuerdo. Los nombres de Román Benítez y Sifre, entre los vivos; de Armaiz, Fernández García y Santiago entre los muertos, representan para la medicina organizada, ejemplo vivo de labor tesonera y altruista, que tuvo el logro de transformar el

panorama de los servicios de la beneficencia pública puertorriqueña.

La dedicación fervorosa, en planos de servicios innovadores y técnicas revolucionarias de la disciplina quirúrgica y que desarrollaran en beneficio de nuestras multitudes hacen que los nombres preclaros de Manuel Figueroa, Lippitt, Jorge del Toro, Perea Fajardo, López Nussa, Vélez López, López Antongiorgi y Díaz García, rebasando las crueles limitaciones de la omisión y el olvido, enaltezcan con el valor de sus enseñanzas de entonces el progreso gigantesco de nuestra cirugía de hoy.

Soslayan también, las dolorosas penumbras de esa omisión y ese olvido por la labor creadora e ingente de las disciplinas rehabilitadoras de la medicina antillana, los nombres ilustres de los Fernández García, Gutiérrez, Muñoz Díaz, López Sicardó, Francisco Seín y Torregrosa.

Cincuenta años han transcurrido desde que la Asociación Médica Puertorriqueña, fiel a las tradiciones de su fundación, enmarcó su vida en el duro laboreo de la medicina organizada.

Asomándonos a través del amplio ventanal, vemos a la Madre Isla, como si se hubiese expandido a lo largo y a lo ancho de sus costas, para dar cabida a dos millones doscientos veinte y cinco mil puertorriqueños.

Se ha creado una escuela de medicina y su estructuración ha respondiendo elocuentemente al justo clamor de la clase médica nativa, que sostuvo con noble intransigencia, los requisitos indispensables para su debida organización y mantenimiento.

Del esfuerzo mancomunado del liderato médico actual y la sobresaliente personalidad médica que se inicia en las aulas de la escuela, surgirá el liderato médico de América y nuestros hombres de ciencia ocuparán las posiciones de vanguardia que le estuvieron vedadas hasta el presente.

No todos podemos vestir la clámide de profesor de nuestra escuela, pero todos debemos estar revestidos con la armadura de valientes y celosos guardianes de su prestigio. Es precisamente en esta etapa de su desenvolvimiento académico, que el afecto vigilante de que hablaba Blanco Fombona, debe ser grano de sal que guarde en sazón, lo que sin él vendría a parar en cuerpo manido.

El panorama ha sufrido alteraciones permanentes, y las legiones productoras que en 1922, tenían un ingreso promedio semanal de escasamente tres pesos, perciben ahora jornales que oscilando entre los dieciseis y veinte, aunque no resuelven definitivamente sus angustias económicas, señalan un ritmo definitivo de progreso.

Las agencias municipales han mejorado considerablemente los salarios de los médicos y la Asociación Médica, aún en medio de sus fallas, ha encauzado en formas permanentes sus empeños de medicina organizada con holgura y con decoro. Numerosas posiciones médicas en pueblos y ciudades están siendo desempeñadas por profesionales extranjeros, con el aparente beneplácito de la clase médica, con la autorización implícita del tribunal de Médicos Examinadores y con el endoso definitivo de la Secretaría de Salud.

El ritmo de la evolución forzada de los últimos cincuenta años, no dió amplio margen al médico nativo para dar rostro a otros problemas, que los del hambre, la pobreza, la enfermedad y la muerte. Al terminar la segunda guerra mundial, los médicos de Puerto Rico que comenzaban a gozar por vez primera de relativa libertad económica, se encontraron combatiendo en vez de la sífilis, la tuberculosis, y el cáncer, nuevos enemigos que se llamaron la incomprensión y la maledicencia organizada, en legiones de vocingleras militancias.

No puede decirse sin embargo que el médico puertorriqueño está haciendo el más mínimo esfuerzo por desacreditar esa conjura organizada, que de manera ostensible debilita los cimientos custodios de nuestra asociación y nuestra clase. Puede afirmarse que la inmensa mayoría vive acogida al piadoso pensar, que esa agresiva campaña es absolutamente gratuita e inmotivada y ha adoptado el hábito peligroso de mirar con desdén sus posibles y mediatas consecuencias.

La clase médica nativa satisfecha de sus actuales éxitos económicos que codician sus compañeros de América creen que la medicina organizada ha de propender a perpetuar este status, sin que medie la labor tesonera y el espíritu de servicio de todos y cada uno de nosotros.

La clámide del médico no otorga inmunidad ni concede prerrogativas en la convivencia de los servidores públicos. Exige por el contrario, en el ejercicio de su ministerio, devoción insospechable hacia el enfermo de todas las esferas de la vida y todos los niveles económicos.

Parodiando a nuestro Brau cabe pensar si urge que sepamos de dónde venimos para que no ignoremos a dónde vamos. Urge tal vez que se defina de una vez y para siempre el alcance final de las leyes que autorizan la importación de médicos extranjeros a Puerto Rico y una vez definido ese alcance, que se logre el ingreso de esos médicos, con residencia permanente en la Isla, a las filas de la medicina organizada.

Cabe pensar sino sería eficaz y conveniente que la clase médica sin abandonar los fines académicos que la han hecho grande, preste intensa atención a los fines de la organización militante que pueda hacerla próspera.

Para los fines de la medicina organizada que significa defensa colectiva con espíritu de gremio; que implica razonable influencia ante las esferas del gobierno; que infiere convivencia y asociación con los médicos extranjeros residentes en Puerto Rico y **requiere pertinentes relaciones de amistad con las ramas legislativas del gobierno**; urge que no vivamos temerosos, ni temperamentalmente distanciados, de las implicaciones naturales y lógicas de una sana política.

Hemos hecho una suscita y escueta narración del balance cultural, científico y humano con que la medicina puertorriqueña ha contribuido al desarrollo de la vida social y económica de la isla, en esta etapa de los últimos cincuenta años. De ese arqueo, conjuntamente con importantísimos aportes, han surgido fallas lamentables, que conviene propender a eliminar. No nos parece sabio, que las nuevas generaciones de médicos se confronten con el panorama desolador de grupos numerosos de médicos puertorriqueños, que no han sentido la urgencia de la asociación, y de médicos extranjeros que no han tenido el logro de dicha convivencia. Urge organizar todos los médicos con residencia permanente en Puerto Rico, en plan de soslayar posibles contingencias de subrepticias organizaciones, entre esos grupos y otros grupos que puedan formarse al calor de nuestra desidia, o ante el impacto de cualquier crisis económica del futuro.

Conociendo de donde venimos marchemos adelante recordando las frases conminatorias de Ernest Hemingway:

"The great thing is to get your work done; and not before, and not too damned much after."

SECCION ADMINISTRATIVA

Cámara de Delegados

El sábado 18 de octubre, a las 3:00 de la tarde, se reunió en sesión extraordinaria la Cámara de Delegados de la Asociación Médica con el propósito de enmendar el Reglamento para reorganizar la Junta de Directores, crear el cargo de presidente electo y crear el Comité de Nominaciones de conformidad con la enmienda introducida a la Constitución en la asamblea extraordinaria celebrada el 21 de septiembre pasado.

La Cámara impartió su aprobación a las enmiendas propuestas, y procedió a designar los restantes cuatro miembros para el Comité de Nominaciones, el cual quedará integrado por los siguientes médicos:

Dr. A. Oliveras-Guerra
Dr. Luis A. Sanjurjo
Dr. Ricardo F. Fernández
Dr. Luis A. Yordán
Dr. Francisco J. Casaldue
Dr. Julio Rodríguez-Olmo
Dr. José A. Peña
Dr. Juan E. Veve
Dr. Francisco A. Márquez
Dr. José A. Seín
Dr. Luis F. Sala
Dr. Miguel A. Valentine
Dr. Rafael A. Gil

El Comité de Nominaciones se reunirá próximamente para empezar su labor de seleccionar y estudiar los distintos candidatos para el cargo de presidente electo de la Asociación.

Servicio Médico Central

El Servicio Médico Central que auspicia la Asociación Médica del Distrito de San Juan empezó a funcionar el día 1ro de octubre y cuenta en la actualidad con 72 suscriptores.

La Asociación Médica del Distrito de San Juan suplica a los suscriptores sigan las siguientes instrucciones, facilitando así el que se les pueda rendir un servicio adecuado tanto a los médicos como al público que hará uso del mismo:

1. Informe a sus pacientes que cuando no puedan localizarle en su casa u oficina llamen al Teléfono 3-2467.
2. Cuando tenga que ausentarse de su casa u oficina tenga la bondad de avisar al teléfono 3-2467 el sitio donde puede ser localizado en caso de que surja alguna emergencia entre sus pacientes.
3. Indique a la persona que esté a cargo del Servicio si desea que sus casos sean referidos a algún otro compañero en caso de que usted no pueda ser localizado.
4. Si tiene alguna sugestión que hacer para que pueda mejorarse este Servicio no vacile en dejárnoslo saber. Sus ideas y consejos serán bien recibidos.

La Asociación Médica del Distrito de San Juan exhorta una vez más

a los médicos residentes en el área metropolitana a que se suscriban a dicho servicio. La cuota a pagar es de \$10.00 trimestrales.

Asamblea Anual

El Comité Científico ya ha completado la organización del programa científico que habrá de regir durante la asamblea anual de la Asociación Médica de Puerto Rico, que se llevará a efecto del 10 al 14 de diciembre próximo.

En la próxima edición del Boletín publicaremos dicho programa.

Curso Postgraduado

Nos complacemos en recordar a la matrícula de la Asociación que del 17 al 21 del próximo mes de noviembre se llevará a efecto en el domicilio de la misma el curso postgraduado que estará a cargo del doctor William S. Middleton, Decano de la Escuela de Medicina de la Universidad de Winsconsin.

Transcribimos a continuación el programa que regirá el mismo:

1. Cardiac Emergencies: Their Recognition and Treatment
2. The Accepted Treatment of Coronary Diseases
3. Recent Advances in Diagnosis and Treatment of Hematological Disorders
4. The Clinical Manifestations of Collagen Disorders
5. Some Clinical Experiences with Adrenocorticotropin and Cortisone.

Asociaciones de Distrito

Nos complacemos en informar a continuación los nombres de los nuevos directores de las siguientes Asociaciones:

Arecibo

J. Rodríguez-Olmo, Pres.
R. Rodríguez-Buxó, Vicepres.
J. R. Valdivieso, Sec.
Julio A. Santos, Tesorero
Delegados: A. H. Susoni
A. Otero López

Mayagüez

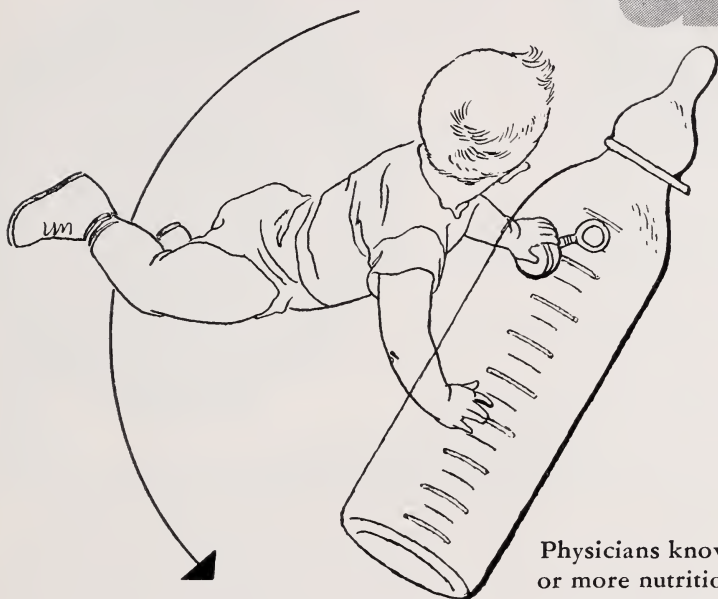
F. J. Casalduc, Pres.
Ramón I. Almodóvar, Vice.
J. Ramírez Ledesma, Sec.
J. Nadal Grau, Tesorero
E. Irizarry Bulls, Delegado
Nayip Fas, Delegado

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Francisco Márquez, Pres.
Susana Igartúa, Vicepres.
Gregorio Igartúa, Sec. y Tes.
Pedro J. Zamora, Delegado

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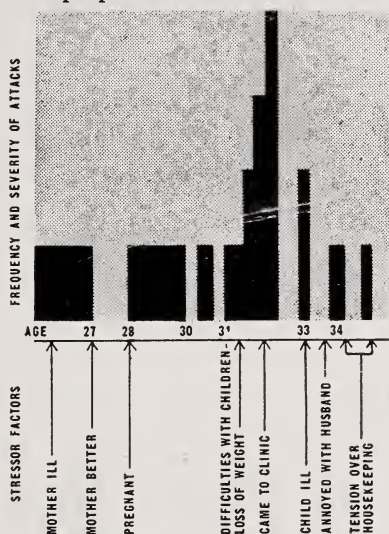
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After Relationship Between Life Stress And Symptoms —
Stevenson, I. G.P. 4: 67 (Dec.) 1951

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¹Cleghorn, R. A. and Graham, B. F.: *Recent Progress in Hormone Research*, Vol. IV, New York, Academic Press, Inc., 1949, p. 323.

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ASAMBLEA ANUAL

DE LA

ASOCIACION MEDICA

DE

PUERTO RICO

DICIEMBRE 10-14 1952

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Ferrous Sulfate, Dried	681 mg.*
Copper (as copper sulfate)	3 mg.
Vitamin B ₁ (thiamine hydrochloride)	15 mg.
Vitamin B ₂ (riboflavin)	6 mg.
Vitamin B ₆ (pyridoxine hydrochloride)	3 mg.
Vitamin B ₁₂ (crystalline)	15 mcg.
Vitamin C (ascorbic acid)	225 mg.
Folic acid	1 mg.
Calcium Pantothenate	3 mg.
Niacinamide	60 mg.
Liver, desiccated, N.F.	525 mg.

**IRONATE
supplies:**

- Iron, plus
- Vitamin B Complex—in significant amount—
- Crystalline Vitamin B₁₂ in substantial dosage
- Copper
- Vitamin C
- Desiccated Liver

*Approximately equivalent to 15 gr. ferrous sulfate, U.S.P. or 204 mg. of elemental iron

**A combination of specific and
adjuvant factors to assure a
prompt and sustained erythro-
poietic response.**

IRONATE®

IRON • VITAMINS • LIVER

SUPPLIED: Bottles of 100 capsules

Wyeth Incorporated, Philadelphia 2, Pa.

**Distribuidores: FRANCISCO N. CASTAGNET
San Juan, Puerto Rico**

HECHOS CLAROS

El 'Bilron' (Sales Férricas de Bilis, Lilly) es un colerético potente; por consiguiente, es muy útil e importante para restablecer y mantener la motilidaad intestinal normal.

En casos de estreñimiento común debido a insuficiencia biliar, la eliminación normal se obtiene corrigiendo la insuficiencia con 'Bilron'. Este producto no contiene ingrediente alguno distinto de los naturales que pueda formar hábito o en alguna otra forma ser dañoso. En casos de estreñimiento colestásico corriente el 'Bilron' es naturalmente eficaz y lógicamente indicado.



CAPSULAS DE BILRON

ELI LILLY PAN-AMERICAN CORPORATION
INDIANAPOLIS 6, INDIANA, E. U. A.

